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Acute Dissociation After 1 Night of Sleep Loss

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Recent research has shown that dissociative symptoms are related to self-reports of deviant sleep experiences. The present study is the 1st to explore whether sleep loss can fuel dissociative symptoms. Twenty-five healthy volunteers were deprived of sleep for 1 night. Sleepiness and dissociative symptoms were assessed every 6 hr. The authors measured both spontaneous dissociative symptoms and dissociative symptoms induced by dot-staring during sensory deprivation. Sleepiness as well as spontaneous and induced dissociative symptoms were stable throughout the day but increased during the night. These findings provide further evidence for a robust relationship between disruptions in sleep patterns and dissociative symptoms.

Keywords: dissociation, sleep deprivation, sleepiness, dissociative disorders

Sleep deprivation is prevalent and has a considerable health, social, and economical impact. For example, the National Sleep Foundation (2005) poll estimated that 29% of adults feel tired at least 3 days a week, and 11% indicate that their sleepiness seriously impacts their daily activities. Also germane to this issue is the finding that rates of work accidents are 7 times higher in individuals who have insomnia than in good sleepers (Leger, Guilleminault, Bader, Levy, & Paillard, 2002). Disruptions in sleep patterns are particularly prominent in some clinical groups: Patients with mood disorders, anxiety disorders, schizophrenia, or borderline personality disorder often have serious sleep dysfunctions (for a review, see Benca, Obermeyer, Thisted, & Gillin, 1992).

A related line of research focuses on the link between dissociative symptoms and sleep. Dissociative symptoms include subjective experiences such as derealization, absorption, and memory complaints. In their mild form, dissociative symptoms are quite common in the general population (Gershuny & Thayer, 1999), yet they are particularly pronounced in diagnostic groups (e.g., individuals with borderline personality disorder, posttraumatic stress disorder [PTSD], schizophrenia, and the dissociative disorders; Holmes et al., 2005; Merckelbach, à Campo, Hardy, & Giesbrecht, 2005). There is an impressive amount of literature on the topic of dissociation; however, researchers in this domain have primarily focused on the alleged traumatic etiology of dissociative experiences (for a review, see Kihlstrom, 2005). Although research on the distal antecedents (i.e., dysfunctional family environment) of

dissociation has revealed clinically relevant findings, little is known about the more proximal mechanisms involved in dissociation. Of interest, a number of recent studies (Giesbrecht, Jongen, Smulders, & Merckelbach, 2006; Giesbrecht & Merckelbach, 2004; Watson, 2001, 2003) have linked dissociative symptoms in undergraduate students to self-reported sleep anomalies, such as flying dreams, hypnopompic imagery, or sensing the presence of someone else. There is also anecdotal evidence that symptoms in patients with depersonalization disorder become worse when they feel tired. Thus, Simeon and Abugel (2006) reported that these patients “often liken it to bad jetlag and feel much worse when they travel across time zones” (p. 210). Similarly, Agargun et al. (2003) emphasized the importance of nightmares in dissociative identity disorder. Taken together, these findings led us to hypothesize that disruptions in the sleep–wake cycle might intensify dissociative symptoms. So far, studies exploring the link between dissociation and sleep pattern disruptions rested entirely on a correlational approach. One inherent limitation of this approach is that it precludes the deduction of causal relations between various variables.

Sleep disturbances can reliably be induced in healthy participants by depriving them of normal sleep. If dissociative experiences are, indeed, fueled by a labile sleep–wake cycle, one would expect sleep loss to increase dissociative symptoms. In the present study, we tested this hypothesis in a sample of healthy undergraduates. We also examined whether sleep loss specifically affects dissociative symptomatology or leads to a more global increase in symptoms. To this end, we measured not only dissociation but also mood fluctuations and the tendency to report hallucinatory experiences.

Method

Participants

Participants were 25 healthy undergraduate students (15 women and 10 men) enrolled at Maastricht University. Their mean age was 19.56 years ($SD = 1.58$; range = 18 to 23 years). Participants

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received a monetary reward (the equivalent of \$150). All participants were nonsmokers. Further exclusion criteria were any kind of sleeping medication, substance misuse or dependence, serious medical disease, or endocrinological disorder. The study was approved by the standing ethical committee of the Faculty of Psychology of Maastricht University, and all participants gave their written informed consent.

Psychometric Instruments

Peritraumatic Dissociative Experiences Questionnaire (PDEQ; Marshall, Orlando, Jaycox, Foy, & Belzberg, 2002). The PDEQ is the most widely used self-report measure of peritraumatic dissociative reactions and consists of eight items. These items quantify the amount of acute dissociation that people experience during a specific event. Respondents are asked to indicate on a 5-point scale (1 = *not at all true*, 5 = *extremely true*) to what extent they experienced particular dissociative symptoms (e.g., "I felt confused or couldn't make sense of what was happening"). In the present study, the Dutch translation of the modified version was used. The PDEQ was always administered in combination with the dot-staring task (described later).

Clinician-Administered Dissociative States Scale (CADSS; Bremner et al., 1998). The CADSS is reliable and valid instrument of state symptoms of dissociation. This scale includes 19 self-report and 8 observer-rated items. Severity of each dissociative symptom can range from 0 (*not present*) to 4 (*extremely present*). Participants were instructed to use the hour preceding the measurement as their reference point. Because of the design of our study, it was not possible to collect accurate ratings for the 8 observer items. Therefore, only the self-report items were administered (see also Morgan et al., 2001). A sample item is "Do you feel as if you are watching the situation as an observer or spectator?"

Stanford Sleepiness Scale (SSS; Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973). The SSS is a measure of subjective sleepiness and consists of a 7-point scale with response options ranging from *feeling active, vital, alert, and wide awake to almost in reverie, sleep onset soon, and lost in struggle to remain awake*. The SSS is widely used in sleep deprivation research (e.g., Babkoff, Caspy, & Mikulincer, 1991).

Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1992). Mood was assessed with the Profile of Mood States—Short Form. The POMS is a self-report measure that is commonly used to index typical and persistent mood reactions to current life situations. Participants indicate on 5-point scales (0 = *not at all*, 4 = *extremely*) to what extent they agree with adjectives (e.g., *annoyed, nervous, angry*) describing their current mood or feelings. The POMS has excellent psychometric properties (see, e.g., Lezak, Howieson, & Loring, 2004; McNair et al., 1992). The present study used a Dutch version of the POMS that has been proven to be both valid and reliable (de Groot, 1991; Wald & Mellenberg, 1990). On basis of the POMS scores, we calculated a general distress composite by summing up all Tension–Anxiety, Depression–Dejection, and Anger–Hostility items. Moreover, the POMS Fatigue–Inertia subscale was used as a concurrent measure of sleepiness.

Cognitive Tasks

Dot-staring task. The dot-staring task has been successfully used by Leonard, Telch, and Harrington (1999) and Miller, Brown, DiNardo, and Barlow (1994) to induce state dissociation in healthy participants. In the present study, participants underwent a 10-min dot-staring task while wearing headphones that dampened all auditory stimulation. This means that all visual and auditory stimulation was minimized temporarily.

White Christmas hallucination (WCH). Participants were instructed that Bing Crosby's famous White Christmas song might be played and that their task was to signal online if they believed they were hearing the song (for an example of this paradigm, see Merckelbach & van de Ven, 2001). Specifically, participants first entered a room in which Bing Crosby's song was playing. Next, they were told that they were going to hear over headphones a tape with white noise for a 3-min period. They then received the following instructions: "the White Christmas song you just heard might be embedded in the white noise below the auditory threshold. If you think or believe that you hear the song clearly, please press the button in front of you. Of course, you may press the button several times if you think that you heard several fragments of the song." Following this, participants were given the headphones, and the tape with white noise was started. The White Christmas song was never presented during the 3-min period. The frequency with which participants pressed the button was recorded.

Procedure

Participants arrived at the lab at 9 a.m. and stayed until 4 p.m. the next day. Thus, participants were not sleep deprived at Day 1, and this allowed us to use participants as their own controls. Participants were tested in groups of 5 and had to stay awake and refrain from coffee during the whole study period. Participants were continuously monitored by at least one experimenter to ensure adherence to instructions. They received standardized meals at noon and 5.30 p.m. on Day 1 and at 12:30 a.m., 7 a.m., and noon on Day 2. Participants were allowed to interact with each other but were discouraged from discussing matters pertaining to the study. Participants were allowed to read, watch DVDs, play games, or surf the Internet. They went for a 30-min walk outside at 6 p.m. on Day 1 and 8 a.m. on Day 2. The rationale of the study was not discussed with participants prior to the debriefing. However, participants were told that they had to stay up for the entire night, until the end of the study, and that every 6 hr they were to complete a number of measures.

Sleepiness and dissociative symptoms were assessed every 6 hr. Sleepiness was measured with the SSS. Present-state dissociative symptoms were quantified with the CADSS. During each measurement session, the SSS and the CADSS were followed by a dot-staring task intended to induce acute dissociation. Acute dissociative experiences were quantified with the PDEQ, which was administered right after each dot-staring session. Next, participants underwent the WCH. The SSS, the CADSS, the dot-staring task, the PDEQ, and the WCH were administered at 9 a.m., 3 p.m., and 9 p.m. on Day 1 and at 3 a.m., 9 a.m., and 3 p.m. on Day 2. Moreover, at 9 a.m., 3 a.m., and 3 p.m. on Day 2, the POMS was administered prior to the SSS and the CADSS. To avoid carry-over

effects, we chose not to counterbalance our measures. That is, we wanted to make sure that our measure of spontaneous dissociative symptoms (i.e., the CADSS) was not confounded by dissociative symptoms elicited during the dot-staring task.

Results

Dissociation and Sleepiness

Figure 1 shows SSS, PDEQ, and CADSS scores throughout the study. These scores were evaluated with analysis of variance with time as a repeated measure. The Greenhouse–Geisser correction was applied to the probability values associated with multiple degrees of freedom repeated measures. Multiple pairwise comparisons were Bonferroni corrected.

Cronbach's alphas were calculated to assess internal consistencies of the PDEQ, the CADSS, and the POMS at different points in time (see Table 1). Note that low Cronbach's alphas during the first part of our study are related to minimal item variability of the measures (i.e., participants scored consistently low). A case in point is the pattern evident for the CADSS. At the first and second test, respectively, only 6 and 8 of the 19 CADSS items displayed nonzero variance. In contrast, on later tests, all or nearly all CADSS items exhibited variability (see Table 1). This is paralleled by the standard deviations of the CADSS (see Table 2). At Tests 1 and 2, standard deviations were 1.46 and 1.36, respectively. However, at later tests, standard deviations increased considerably, with a standard deviation of 7.95 at the final test, which is a 4.45-fold increase compared with the first test. Other measures

followed a similar pattern with substantially higher standard deviations and Cronbach's alphas at later tests relative to earlier tests. We note in passing that at the final tests, Cronbach's alphas are high. Thus, it is certainly not the case that after sleep loss, participants filled out questionnaires more haphazardly because of, for example, poor attention or concentration.

We investigated whether sleepiness, as measured with the SSS, increased over time. This analysis revealed a significant main effect of time, $F(3.62, 24) = 27.16, p < .01, \eta^2 = .53$. Pairwise comparisons between adjacent time points indicated that this effect was due to sleepiness increases between 9 p.m. and 3 a.m., $t(24) = 2.14, p < .05$. Using a similar analytic approach for CADSS and PDEQ scores, we tested whether dissociative levels increased as a function of sleep deprivation. Indeed, both CADSS scores, $F(1.58, 24) = 7.86, p < .01, \eta^2 = .25$, and PDEQ scores, $F(1.59, 24) = 18.64, p < .01, \eta^2 = .44$, increased over time. Pairwise comparisons between adjacent time points showed that this increase manifested itself both between 9 p.m. and 3 a.m.—CADSS, $t(24) = 3.02, p < .05$; PDEQ, $t(24) = 3.66, p < .01$ —and between 3 a.m. and 9 a.m.—CADSS, $t(24) = 3.36, p < .05$; PDEQ, $t(24) = 4.94, p < .01$.

Table 3 presents nonparametric zero-order intercorrelations between SSS, CADSS, PDEQ, and WCH scores at the different test sessions. As can be seen, for most sessions after 3 a.m. at Day 2, dissociation levels, as measured by the CADSS or the PDEQ, were significantly related to sleepiness, as measured by the SSS. During these test sessions, CADSS and PDEQ correlated with each other, indicating that they tap related constructs. However, correlations

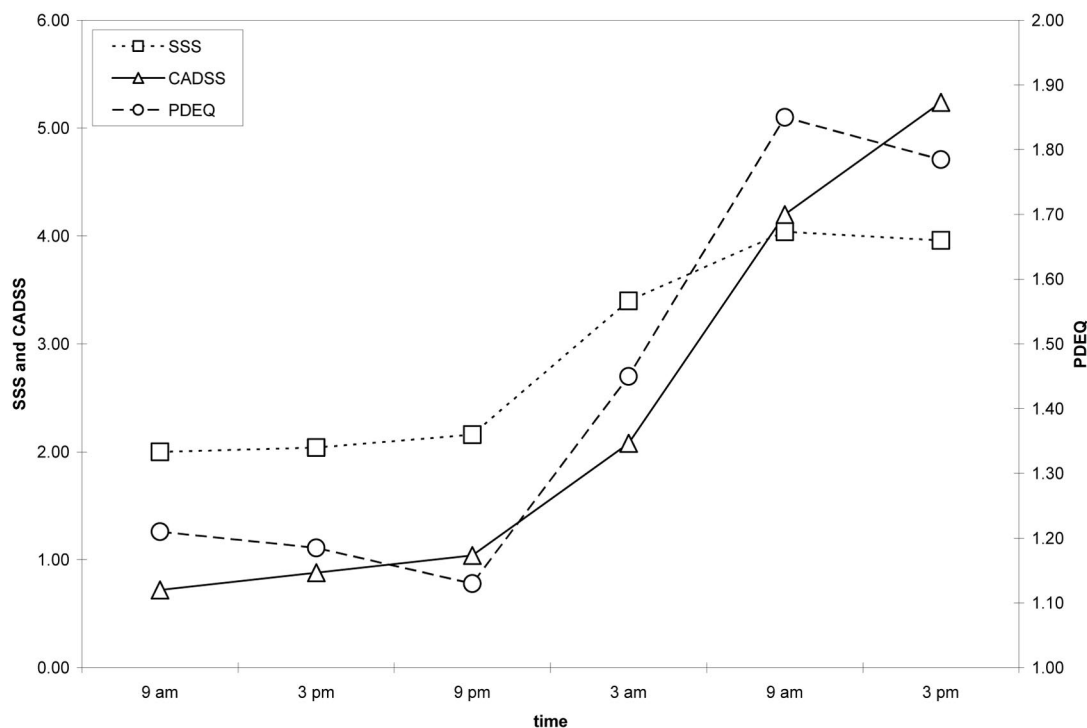


Figure 1. Scores on the Stanford Sleepiness Scale (SSS), the Clinician-Administered Dissociative States Scale (CADSS), and the Peritraumatic Dissociative Experiences Questionnaire (PDEQ) for an undergraduate sample ($N = 25$) at different testing times.

Table 1
Internal Consistency and Item Variability of Study Measures at Different Tests for an Undergraduate Sample ($N = 25$)

Measure	Day 1						Day 2					
	9 a.m.		3 p.m.		9 p.m.		3 a.m.		9 a.m.		3 p.m.	
	α	Item variability ^a	α	Item variability ^a	α	Item variability ^a	α	Item variability ^a	α	Item variability ^a	α	Item variability ^a
CADSS (19 items)	.42	6	.29	8	.79	18	.80	18	.84	18	.94	19
PDEQ (8 items)	.27	6	.26	8	.42	7	.85	7	.88	8	.84	8
POMS FI (6 items)	.76	6					.88	6			.92	6
POMS GD (18 items)	.40	13					.30	11			.86	17

Note. CADSS = Clinician-Administered Dissociative States Scale; PDEQ = Peritraumatic Dissociative Experiences Questionnaire; POMS FI = Profile of Mood States Fatigue–Inertia subscale; POMS GD = Profile of Mood States general distress composite.

^a Table values for item variability are the number of items for a given test without zero variance.

between the dissociation measures were not consistent, that is, they fell short of significance at Day 1 but were significant at Day 2. We believe that this has to do with the low variability of some of our measures, especially on Day 1 (see Table 2). To investigate this, we collapsed data across all time points and then again calculated correlations (see the lower part of Table 3). Evidently, this approach greatly increases the range of the measures. Of interest, the resulting correlational pattern is much more straightforward. In line with our Day 2 findings, the CADSS and the PDEQ overlapped considerably, as did our two measures of fatigue (i.e., the SSS and the POMS FI). Moreover, both measures of fatigue were related to dissociation scores with correlations ranging from .51 to .43.

WCH

Table 2 presents mean frequencies (and standard deviations) with which participants reported hearing fragments from the White Christmas song. To investigate whether this frequency changed after sleep loss, we conducted a repeated measures analysis of variance with time as the repeated measure. This analysis did not reveal a significant effect of time, $F(2.87, 24) = 1.53, p > .05, \eta^2 = .06$. Moreover, correlational analyses with data collapsed across all time points showed that WCH scores were weakly related to the CADSS and unrelated to the PDEQ.

POMS

Table 2 also shows POMS Fatigue–Inertia and general distress composite scores throughout the study. We examined whether fatigue increased over time. As was the case for the SSS index, this yielded a significant main effect of time, $F(1.41, 24) = 40.91, p < .01, \eta^2 = .63$. Pairwise comparisons between adjacent time points indicated that this effect was due to Fatigue–Inertia increases both between 9 a.m. and 3 a.m., $t(24) = 3.47, p < .01$, and between 3 a.m. and 3 p.m., $t(24) = 5.92, p < .01$. Furthermore, we tested whether general distress, as measured by the POMS general distress composite, increased over time. This analysis revealed a significant main effect of time, $F(1.29, 24) = 8.65, p < .01, \eta^2 = .27$. Pairwise comparisons between adjacent time points indicated that this effect was due to distress increases between 3 a.m. and 3 p.m., $t(24) = 2.92, p < .01$. Thus, POMS general distress scores increased at a later point in time than dissociation scores.

To further explore the possibility that acute dissociation levels might be mediated (Baron & Kenny, 1986) by general distress rather than sleepiness per se, we conducted two stepwise regression analyses with CADSS and PDEQ indices of acute dissociation during the final test session (i.e., at maximal sleep loss) as dependent variables. Gender, age, POMS Fatigue–Inertia, and POMS general distress, measured at the same point in time, functioned as predictors. These analyses indicated that the POMS Fatigue–

Table 2
Mean Scores and Standard Deviations of Study Measures for an Undergraduate Sample ($N = 25$) at Different Test Sessions

Measure	Day 1						Day 2					
	9 a.m.		3 p.m.		9 p.m.		3 a.m.		9 a.m.		3 p.m.	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
SSS	2.00	0.91	2.04	0.89	2.16	0.80	3.40	0.87	4.04	1.37	3.96	1.40
CADSS	0.72	1.46	0.88	1.36	1.04	2.24	2.08	2.97	4.20	4.60	5.24	7.95
PDEQ	1.21	0.20	1.19	0.16	1.13	0.16	1.45	0.47	1.85	0.73	1.79	0.69
WCH	1.12	1.56	1.28	1.95	1.12	1.56	1.16	1.52	1.24	1.79	0.60	0.96
POMS FI	2.28	2.39					4.56	3.83			11.80	6.64
POMS GD	5.28	2.62					5.80	2.43			8.60	5.66

Note. SSS = Stanford Sleepiness Scale; CADSS = Clinician-Administered Dissociative States Scale; PDEQ = Peritraumatic Dissociative Experiences Questionnaire; WCH = White Christmas Hallucination; POMS FI = Profile of Mood States Fatigue–Inertia subscale; POMS GD = Profile of Mood States general distress composite.

Table 3

Spearman Intercorrelations Between the Study Measures in an Undergraduate Sample (N = 25) at Different Points and Collapsed Across All Points In Time

Measure	Day 1, 9 a.m.					Day 1, 3 p.m.			Day 1, 9 p.m.				
	SSS	CADSS	PDEQ	WCH	POMS FI	SSS	CADSS	PDEQ	SSS	CADSS	PDEQ	WCH	POMS FI
Day 1													
SSS	—					—			—				
CADSS	0.19	—				0.20	—		0.15	—			
PDEQ	-0.12	-0.12	—			-0.10	0.15	—	0.13	0.33	—		
WCH	-0.08	0.25	0.18	—		0.04	0.23	0.00	0.19	0.31	-0.14		
POMS FI	0.41*	0.15	-0.51*	0.18	—	—	—	—	—	—	—		
POMS GD	0.32	-0.24	0.10	-0.03	0.23	—	—	—	—	—	—		
Day 2													
SSS	—					—			—				
CADSS	0.32	—				0.49*	—		0.53*	—			
PDEQ	0.40*	0.53*	—			0.54*	0.75*	—	0.31	0.48*	—		
WCH	-0.13	0.35	0.41*	—		-0.24	-0.01	-0.01	0.08	0.41*	0.20	—	
POMS FI	0.30	0.49*	0.22	0.19	—	—	—	—	0.47*	0.56*	0.48*	0.08	—
POMS GD	-0.26	-0.28	-0.15	-0.25	-0.07	—	—	—	0.44*	0.31	0.25	0.24	0.49*
Combined ^a													
SSS	—					—			—				
CADSS		0.51*			—								
PDEQ		0.51*			0.53*			—					
WCH		-0.03			0.21*			0.07			—		
POMS FI		0.61*			0.48*			0.43*			0.07		—
POMS GD		0.25*			0.05			0.24*			-0.07		0.36*

Note. SSS = Stanford Sleepiness Scale; CADSS = Clinician-Administered Dissociative States Scale; PDEQ = Peritraumatic Dissociative Experiences Questionnaire; WCH = White Christmas Hallucination; POMS FI = Profile of Mood States Fatigue-Inertia subscale; POMS GD = Profile of Mood States general distress composite.

^a Between all measurements across testing.

* $p \leq .05$.

Inertia subscale accounted for 42% of the variance in CADSS scores ($B = 0.78$, $\beta = .65$), $t(24) = 4.11$, $p < .01$, and 20% of the variance in PDEQ scores ($B = 0.05$, $\beta = .44$), $t(24) = 4.60$, $p < .05$. All other predictors were not significant. Thus, the effect of sleep deprivation on dissociation was not mediated by general distress.

Discussion

To the best of our knowledge, the present study is the first indicating that sleep loss intensifies dissociative symptoms. This was found to be true for both spontaneous dissociative symptoms as measured by the CADSS and symptoms induced by dot-staring and tapped by the PDEQ. In addition, mood, as indexed by our POMS general distress composite, worsened but exhibited a different temporal pattern than dissociation, whereas auditory hallucinatory experiences induced by the WCH did not increase after sleep loss.

Since the pioneering study of Orne and Scheibe (1964) on sensory deprivation, it has been known that designs such as the present one are susceptible to demand characteristics. There are good grounds for believing that individuals who score high on

dissociation are especially sensitive to such tendencies (Merckelbach & Jelicic, 2004). However, there are three reasons why our findings cannot be solely accounted for by demand characteristics. To begin with, dissociative symptoms followed a specific temporal pattern. That is, they remained stable during the day (i.e., from 9 a.m. to 9 p.m.) and only increased in the evening and during the night. This is in line with the findings of Babkoff and coworkers (1991), who showed that sleepiness after sleep loss underlies strong circadian oscillations. More specifically, these authors showed that sleepiness increases during the night but then recovers slightly during daytime. In the present study, increases in dissociative symptomatology appeared to follow such circadian oscillations in sleepiness. However, our study relied on only 1 night of sleep deprivation. Future studies investigating sensitivity of dissociative symptoms to circadian oscillations after sleep deprivation should preferably use longer periods of sleep loss.

A second reason why our findings cannot be fully accounted for in terms of demand is that scores on both dissociation measures increased in parallel. Meanwhile, our participants did not fall prey more often to the WCH after sleep loss. If our dissociation findings were entirely driven by demand characteristics, one would expect

that this would also affect hallucinatory reports on the WCH. One would then expect increasing frequencies of hallucinatory reports over time, but we did not find such a pattern. Moreover, when data were collapsed across all time points, correlations between the WCH and the CADSS and the PDEQ remained weak at best. Of interest, items tapping hallucination-like experiences are often included in scales measuring hypnotic suggestibility, but studies show that correlations between trait dissociation and hypnotic suggestibility are on the order of only .14 (Kirsch & Lynn, 1998). Likewise, in our study, correlations across all time points between the WCH and the PDEQ and the CADSS were .21 and .07, respectively.

A third reason why the present results cannot be satisfactorily explained in terms of a demand bias to express negative or unusual experiences is that mood changes did not parallel the increase in dissociative symptoms. In sum, then, there are good empirical reasons to rule out demand characteristics as the principal driving force behind our findings. However, as our study did not include a control condition of participants who simulated being sleep deprived, we cannot definitely rule out the possibility that demand characteristics played some role in shaping the results.

A number of previous studies have noted that eating disorders (Valdiserri & Kihlstrom, 1995), trauma (Mulder, Beautrais, Joyce, & Fergusson, 1998), and thought intrusions (Spinhoven & van der Does, 1999) are linked to dissociation in an indirect way, that is, through the operation of third variables. Thus, one could speculate that sleep loss does not influence dissociative symptomatology directly but rather has a detrimental influence on mood, which in turn might lead to a heightened predisposition to dissociate or simply increase participants' tendency to say eccentric things about themselves. However, our findings do not support this line of reasoning. We found that dissociative symptoms increase prior to mood deteriorations. Furthermore, we found that even after maximum sleep loss, fatigue rather than mood deteriorations predict dissociative tendencies. Unfortunately, we administered mood measures less frequently than dissociation and sleepiness measures, which makes it difficult to compare the precise trajectories of these variables.

Because our participants were healthy undergraduates with homogeneous (i.e., low) initial dissociation levels, our study cannot elucidate whether sleep loss actually triggers dissociation or works as a catalyst, increasing preexisting dissociative tendencies. Therefore, it would be helpful if future sleep-deprivation studies would look at the relationship between traitlike dissociative tendencies and increases in dissociative symptomatology in more diverse and larger samples. Indeed, sample size is important, as one of the problems of the current study is that its limited sample hampers interpretation of nonsignificant correlations at various points in time. It would also be informative if future studies would systematically sample depression and schizotypy. Depression has been shown to be related to insomnia (Tsuno, Besset, & Ritchie, 2005), and schizotypy is intimately linked to both nightmares (e.g., Watson, 2001) and dissociation (Merckelbach & Giesbrecht, 2006).

The effect of sleep loss on dissociative symptoms that we found in the present study is likely to be an underestimation, as our participants were healthy undergraduates. This group might be less affected by sleep deprivation than clinical groups. Older individuals require less sleep than younger ones. Therefore, the effect of a single night of sleep deprivation on dissociation might be dif-

ferent in various age groups. Nevertheless, the relatively high incidence of sleep dysfunctions (National Sleep Foundation, 2005) highlights the relevance of our findings, particularly because the amount of sleep loss that we induced in the present study is regularly encountered in the general population.

Our findings nicely fit with the notion that disruptions in circadian rhythms affect wakefulness and arousal and have detrimental effects on memory and attentional control (see also Simeon & Abugel, 2006; Watson, 2001). Thus, sleepiness might contribute to the attentional deficits that are typically found in undergraduates high on dissociation (Giesbrecht, Merckelbach, Geraerts, & Smeets, 2004) and in patients with dissociative disorders (Guralnik, Schmeidler, & Simeon, 2000; Simeon & Abugel, 2006). Germane to this issue are studies showing that dissociation and depersonalization symptoms are related to lowered levels of urinary norepinephrine (Simeon, 2004), a neurotransmitter regulating arousal and alertness. An additional consequence of disruptions in the sleep-wake cycle of individuals high on dissociation might be the intrusion of sleep phenomena (e.g., dreamlike experiences) into waking consciousness, resulting in feelings of depersonalization and derealization (Watson, 2001).

The finding that sleep loss increases acute dissociative symptoms might have implications for groups that are regularly sleep deprived, such as medical interns and long-shift workers. Sleep-deprived people have a higher risk of becoming involved in accidents (e.g., Leger et al., 2002). This accident proneness is commonly ascribed to poor attention and concentration due to sleep loss (Jewett, Dijk, Kronauer, & Dinges, 1999). However, our findings raise the possibility that in sleep deprived people, acute dissociative symptoms might interfere with conscious cognitive control, which in turn may make these people accident prone. Tentative evidence for this comes from studies showing a robust overlap between traitlike dissociative tendencies and scores on the Cognitive Failures Questionnaire (CFQ; Merckelbach, Horselenberg, & Schmidt, 2002; Merckelbach, Muris, & Rassin, 1999). The CFQ measures the frequency of everyday cognitive failures, such as forgetting names, missing signs on the road, or being distracted. Individuals who score high on the CFQ are also more likely to become involved in accidents (Wallace & Vodanovich, 2003). Clearly, the links between sleep loss, acute dissociative symptoms, and accident proneness merit further research.

The present findings may also be helpful in understanding why there is such a robust connection between dissociation and memory commission errors. A typical feature of the cognitive architecture of people high on dissociation is that they tend to produce pseudomemories (i.e., false alarms, commission errors) on memory tasks (Candel, Merckelbach, & Kuijpers, 2003; Giesbrecht, Geraerts, & Merckelbach, 2007; Merckelbach, Zeles, van Bergen, & Giesbrecht, 2007). Thus, it may well be that the dreamlike intrusions into waking state that are typical of dissociation interfere with memory performance in such a way that commission errors occur. The present study did not include any memory measures, yet we do think that the link between dissociation, memory commission errors, and sleep loss warrants further study.

In sum, although the present study is a long way from offering direct therapeutically relevant insights, it does highlight the relevance of (disruptions in) sleep patterns to dissociative symptoms and dissociative disorders. Our findings require replication in clinically relevant groups, and if such replications are successful,

the question would arise of whether patients with dissociation may profit from therapeutic approaches directed at normalizing sleep patterns and reducing nightmares. Taken together, the present and earlier findings (Giesbrecht et al., 2006; Giesbrecht & Merckelbach, 2004, 2006; Watson, 2001) suggest that the study of how sleep patterns relate to dissociative symptoms is a fruitful research area that might eventually lead to new therapeutic approaches in the treatment of the dissociative disorders and other conditions that are characterized by heightened levels of dissociation (e.g., PTSD).

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